to be defined. But there is much at stake in all of this for the future of the medical profession. If it is to play its proper role, the profession itself should earn recognition as "physician to society" in all matters pertaining to health and health care, in much the same sense that a doctor is recognized as physician to a patient, but it will surely take new concepts and better performance in CME to bring all this about.

MSMW

Radiocontrast Nephrotoxicity

ACUTE RENAL FAILURE occurs frequently in the contemporary practice of medicine. Hou and collaborators studied prospectively 2,216 in-hospital patients and found that some degree of acute renal insufficiency developed in 4.9%.[1] The incidence of acute renal failure approaches 15% to 50% in selected clinical settings such as intensive care unit admission, serious trauma and burns, complicated open heart and abdominal aortic aneurysm operations and following aminoglycoside therapy.[2] Despite significant advances in the care of patients with acute renal failure and the ready availability of dialysis, current mortality rates are 15% to 25% for nonoliguric and 30% to 80% for oliguric acute renal failure.[3] The high frequency of occurrence and significant mortality of acute renal failure demand that attention be directed to preventive measures.

It is noteworthy that 15% to 25% of all cases of acute renal failure can be attributed to commonly used therapeutic agents.[2] Several factors underlie renal vulnerability to toxic injury. Normal renal function requires the interaction of both vascular and tubular elements and injury to either can lead to renal failure. The kidney receives 25% of the cardiac output and is exposed to high concentrations of blood-borne substances. The ability of the kidney to concentrate glomerular filtrate severalfold plus renal tubular transport processes exposes renal cells to even higher concentrations of potential toxins. Numerous enzymatic and metabolic pathways are operative within the kidney that may be particularly susceptible to nephrotoxic injury. Because of these factors, therapeutic agents such as immunosuppressants (cyclosporine), antimicrobials (aminoglycosides, amphotericin B), heavy metals (cisplatin) and anti-inflammatory drugs (nonsteroidals) are often implicated as causes of acute renal failure.[2]

In this issue of the journal, Misson and Cutler provide a comprehensive overview of acute renal failure following the diagnostic use of radiocontrast agents. Four "take-home" messages of this excellent review are as follows: (1) radiocontrast agents appear remarkably safe in healthy persons. In the presence of normal renal function, radiocontrast agents result in a less than 1% to 2% incidence of nephrotoxicity. (2) Patients with renal insufficiency (especially patients with diabetic nephropathy) are at high risk for radiocontrast nephrotoxic effects. A significant increment in serum creatinine concentration occurs in from 10% to 90% of azotemic patients. (3) Most patients with radiocontrast-associated acute renal failure experience a mild, transient increase in serum creatinine concentration. However, in 20% to 30% of patients, the increase in serum creatinine concentration is more pronounced or not reversible (or both). (4) The mechanism(s) underlying radiocontrast nephrotoxicity are unclear. Evidence supporting abnormalities of both renal vascular and tubular function is available.

Unfortunately, radiocontrast agents are often felt to be indicated in the assessment of patients at high risk for the development of acute renal failure. What is the best approach in such patients? Avoiding unnecessary contrast procedures is the first step. Other diagnostic procedures may provide the needed information. For example, excretory urography is often used to assess kidney size and the presence or absence of obstructive uropathy in patients with renal failure. Recent studies indicate that ultrasonography in experienced hands is a sensitive method for determining renal size and excluding obstructive uropathy.[4] Other preventive measures suggested by Misson and Cutler, such as avoiding volume depletion during preparation, avoiding the concomitant use of other nephrotoxins, using the smallest possible dose of contrast agent and maintaining a three-day interval between repetitive contrast exposures, are practical but unproven suggestions.

Are additional preventive measures indicated in a high-risk patient? No large prospective controlled studies are available to answer this question. However, retrospective studies suggest that administering 1.5 liters of a solution of 0.45% saline attenuates the effect of intravenous urography to elevate serum creatinine concentration in patients with chronic renal failure.[5,6] In addition, an uncontrolled prospective study of more than 500 patients suggests that a comparable or slightly greater degree of volume expansion results in a low frequency of acute renal failure following angiography even in high-risk patients.[7] Together, these observations support the recommendations of Misson and Cutler that until better data are available, patients with a serum creatinine concentration of more than 2 mg per dl (more than 1.5 mg per dl in cases of diabetic nephropathy) receive volume expansion (1.5 to 2.5 liters) before and during contrast exposure. What should be done in high-risk patients who are not suitable candidates for moderate volume expansion? A preliminary report of a prospective controlled trial of 27 patients suggests that 50 grams of mannitol in 180 ml of 5% dextrose solution given within 45 minutes of radiocontrast exposure attenuates the increment in serum creatinine.[8] Such therapy is usually well tolerated clinically. There are no clinical studies to support the proposed prophylactic use of potent diuretic agents, such as furosemide, in patients at high risk for radiocontrast nephrotoxicity.[9]

What does the future hold? Several laboratories are actively working on the pathophysiology of radiocontrast-associated acute renal failure in animal models. Such studies may lead to a more scientific approach to prophylactic therapy. As noted by Misson and Cutler, efforts are also under way to develop less nephrotoxic radiocontrast agents. Finally, newer visualization procedures which do not require radiocontrast exposure, such as nuclear magnetic resonance, may provide both physiologic and anatomic information. In the meantime, avoidance of unnecessary procedures and prophylactic therapy in high-risk patients appears to be the best course to follow.

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Health Care Dollars to Other Pockets

IT SEEMS REASONABLE to ask where all the health care profits are coming from and where they are going. There is considerable reason to believe that a large portion of these profits is the result of restrictions on those who provide and those who receive the services. The premise of the rhetoric we hear is that the profit motive will increase efficiency, reduce costs and eliminate the "fat"—and do all this without curtailing access or quality of care. The incentive for profit and the opportunity for competition is supposed to bring all this about, even in what may already be the most regulated health care system in the world.

There have always been profits in the health care field. The pharmaceutical industry has always done well and has had the incentives necessary to develop the wherewithal for much of the progress in patient care that has occurred. And some physicians have done very well indeed. Recently a growing number of hospitals and other health care institutions are being operated for profit. In the past, good hospitals, good skilled nursing facilities, good nursing homes and good home care agencies have not been considered very profitable operations. In fact, many, if not most, were operated "not for profit" and any "profits" were plowed back into the institution.

But now the health care system is being leached for dollars in ways it has seldom been, at least within recent memory. In the name of business efficiency, and to deal with the almost impossible complexities of government regulation, costly administrative hierarchies (with their accompanying organizational bureaucracies) are developing in hospitals and health care institutions that leach health care dollars for salaries and benefits for their ever-expanding management structures. The advertising, marketing and amenities stimulated by a growing competition for patients, pad the pocketbooks of many who actually contribute little if anything to health care. And the current orgy of malpractice and other litigation, so profitable to trial lawyers and others who thrive on litigation, adds new and often substantial costs to physicians' fees and the cost of a day's stay in hospital.

The public rhetoric continues to say—as it has for some time—that our goal is to have equal access for all to good quality medical care (although the *right* to care has been considerably muted), but that the cost of this has now become unacceptable. Yes, one may reasonably ask where all the profits in health care are coming from and where are they going. It is now becoming increasingly clear that in large measure they must be coming from restrictions on the providers of care and limitations on the options of patients who are the consumers of care, and both have had precious little to say about what is being done to them. One can only wonder if the goal should not be to get more of the needed health care from the admittedly scarce health care dollars, rather than to encourage all the competition, regulation and profiteering that divert so many needed health care dollars to other pockets.

MSMW

Adult Still's Disease—Implications of a New Syndrome

IN 1897 George Frederick Still felt it important to emphasize that the syndrome which came to bear his name be distinguished from rheumatoid arthritis in adults.[1] Almost 90 years later, we must decide if we should recognize an adult form of Still's disease as a separate nosologic entity. Citing the importance of the diagnostic, therapeutic and prognostic implications of recognizing a rare condition, several authors[2-4] have advocated the acceptance of the term adult Still's disease since it was coined by Bywaters in 1966.[5]

In this issue of the journal and elsewhere, Larson further supports this position by extensively reviewing the clinical features of the majority of reported cases of adult Still's disease and of 17 patients followed at the University of Washington.[4] In favor of recognizing adult Still's disease as a distinct syndrome is the remarkably consistent description of the clinical picture, with high fever, intense arthralgias or arthritis and a characteristic salmon-colored rash as prominent findings. Characteristically, the disease spares the metacarpophalangeal joints, a feature quite different from what is usually seen in rheumatoid arthritis. Standard serologic studies, such as antinuclear antibody and rheumatoid factor, are negative. An unusual ankylosing of the carpal bones occurs chronically; this is quite unlike more familiar rheumatic diseases, such as active rheumatoid arthritis in adults, mixed connective tissue disease or systemic lupus erythematosus.

But is this evidence enough? After all, it also is clear that the etiology is uncertain, no serologic tests are diagnostic[3] and, despite the existence of characteristic findings, in most cases the diagnosis is one of exclusion. Furthermore, probably because adult Still's disease is relatively rare, few longitudinal studies have been done. We are already learning that the disease is not as benign as once thought, with chronic arthritic changes developing in some patients. In view of this, can we be certain that patients with adult Still's disease will not eventually be found to be a subset of populations with another disease? This situation is reminiscent of what has occurred in mixed connective tissue disease where the diagnosis often rests mainly on a high titer of antiribonucleoprotein antibody. Some rheumatologists doubt the existence of mixed connective tissue disease as a distinct clinical entity; they point out that in a proportion of such patients followed for a decade or more, their disease appears to have evolved into typical systemic sclerosis or another connective tissue disorder.

Nonetheless, several standard sources of information, including *Harrison's Textbook of Medicine*, currently accept adult Still's disease as a separate disorder. The American Rheumatism Association also acknowledges its existence,[6]